What is claimed is:

Claim 1. A method for testing a candidate drug for treatment or prevention of age-related macular degeneration (AMD) comprising administering the candidate drug to a Ccl2-deficient knockout mouse, a Ccr2-deficient knockout mouse and/or a Ccl2-deficient/Ccr2-deficient dual knockout mouse and analyzing at least one eye for development or regression of drusen and/or lipofuscin accumulation therein, for affect of the candidate drug on Bruch's membrane, affect on retinal degeneration, in at least one eye, and/or affect on choroidal neovascularization of at least one eye.

Claim 2. The method of claim 1 wherein the candidate drug is nucleic acid.

Claim 3. The method of claim 2 wherein the nucleic acid comprises a viral vector encoding wild-type Ccl2.

Claim 4. The method of claim 1 wherein the nucleic acid comprises a viral vector encoding wild type Ccr2.

Claim5. The method of claim 1 wherein analyzing the at least one eye comprises determining amount and type of drusen or lipofuscin, extent of retinal degeneration, or neovascularization developed therein.

Claim 6. The method according to claim 1 wherein the at least one eye is analyzed by ophthalmoscopy, angiography, histopathology or a combination thereof.

Claim 7. The method of claim 1 wherein the candidate drug is administered to the mouse orally, intravenously, intraperitoneally, intravitreously, transsclerally or topically.

Claim 8. The method of claim 7 wherein the candidate drug is administered topically to at least one eye of the mouse.

Claim9. The method of claim 1 wherein the candidate drug is a pharmaceutical compound, small molecule, peptide, antibody, antibody fragment, aptamer or nucleic acid.

Claim 10. The method of claim 9 wherein the nucleic acid is an oligonucleotide or polynucleotide in either the sense or antisense orientation or an aptamer.

Claim 11. A method of screening a test compound for potential utility for treatment of age-related macular degeneration, comprising:

(a) providing a mouse comprising a disrupted Ccl2 and/or CCR2 gene, wherein the mouse is homozygous for the disrupted gene or genes, and wherein the mouse exhibits drusenand/or lipofuscin deposits, retinal degeneration, and/or choroidal neovascularization in at least one eye at about nine to twenty-four months of age compared to a wild-type mouse that does not have the disrupted gene;

(b) administering the test compound to the mouse;

(c) determining the effect of the test compound on drusen, lipofuscin deposition, retinal degeneration, or choroidal neovascularization in at least one eye of the mouse; and

(d) correlating the effect of the test compound on drusen, lipofuscin accumulation, retinal degeneration, and/or choroidal neovascularization with a potential utility to treat age-related macular degeneration.

Claim 12. The method of claim 11 wherein the test compound is administered topically to at least one eye of the mouse.

Claim 13. The method of claim 11 wherein the candidate drug is nucleic acid.

Claim14. The method of claim 11 wherein the nucleic acid comprises a viral vector encoding wild-type Ccl2.

Claim 15. The method of claim 11 wherein the nucleic acid comprises a viral vector encoding wild type Ccr2.

Claim16. The method of claim 11 wherein analyzing the at least one eye comprises determining amount and type of drusen or lipofuscin, retinal degeneration or neovascularization developed therein.

Claim 17. The method according to claim 11 wherein the at least one eye is analyzed by ophthalmoscopy, angiography, histopathology, mass spectometry or a combination thereof.

Claim 18. The method of claim 11 wherein the candidate drug is administered to the mouse orally, intravenously, intraperitoneally, intravitreously, transsclerally or topically.

Claim 19. The method of claim 18 wherein the candidate drug is administered topically to at least one eye of the mouse.

Claim20. The method of claim 11 wherein the candidate drug is a pharmaceutical compound, small molecule, peptide, antibody, antibody fragment, aptamer or nucleic acid.

Claim 21. The method of claim 13 wherein the nucleic acid is an oligonucleotide or polynucleotide in either the sense or antisense orientation or an aptamer.

Claim 22. A Ccl2-deficient/CCR2-deficient dual knockout mouse.

Claim 23. The method of claim 7 wherein the candidate drug is administered intraviteously by injection or by sustained delivery implant, to at least one eye of the mouse.

Claim 24. The method of claim 7 wherein the candidate drug is administered transsclerally to at least one eye of the mouse.

Claim 25. The method of claim 11 wherein the test compound is administered intravitreously by injection or by sustained delivery implant, to at least one eye of the mouse.

Claim 26. The method of claim 11 wherein the test compound is administered transsclerally to at least one eye of the mouse.

Claim 27. The method of claim 18 wherein the candidate drug is administered intravitreously by injection or by sustained delivery implant to at least one eye of the mouse.

Claim 28. The method of claim 18 wherein the candidate drug is administered transsclerally to at least one eye of the mouse.

Claim 29. A method of monitoring the expression of a Ccr2 gene in at least one eye of a Ccr2-/- mouse comprising

(1) introducing a plurality of stem cells obtained from a wild type mouse into the Ccr2-/- mouse to obtain a transplanted mouse, wherein said stem cells express wild type Ccr2;

(2) observing at least one eye of the transplanted mouse for the effect of the wild type Ccr2 gene expression on drusen or lipofuscin deposition, retinal degeneration, or choroidal neovascularization in at least one eye of the transplanted mouse.

Claim 30. The method of claim 29 wherein the wild type Ccr2 gene is introduced by adoptive transfer or following bone marrow ablation of the Ccr2-/- mouse.

Claim 31. A method of monitoring the effects of expression of a Ccl2 gene in at least one eye of a Ccl2-/- mouse comprising

(1) introducing a plurality of stem cells obtained from a wild type mouse into the Ccl2-/- mouse to obtain a transplanted mouse, wherein said stem cells express wild type Ccl2;

(2) observing at least one eye of the transplanted mouse for the effect of the wild type Ccr2 gene expression on drusen or lipofuscin deposition, retinal degeneration, or choroidal neovascularization in at least one eye of the transplanted mouse.

Claim 32. The method of claim 31 wherein the wild type Ccl2 gene is introduced by adoptive transfer or following bone marrow ablation of the Ccl2-/- mouse.

Claim 33. A method of monitoring the effects of expression of a Ccl2 gene, Ccr2 gene or both in at least one eye of a Ccl2 deficient, Ccr2 deficient mouse comprising

(1) introducing a plurality of stem cells obtained from a wild type mouse into the Ccl2 deficient, Ccr2 deficient mouse to obtain a transplanted mouse, wherein said stem cells express wild type Ccl2 and Ccr2;

(2) observing at least one eye of the transplanted mouse for the effect of the wild type Ccl2 and/or Ccr2 gene expression on drusen or lipofuscin deposition, retinal degeneration, or choroidal neovascularization in at least one eye of the transplanted mouse.

Claim 34. A method of identifying mutations in the Ccl2 gene, Ccr2 gene, Ccl2 promoter, Ccr2 promoter, Ccl2 enhancer, or combinations thereof comprising

(1) obtaining an AMD DNA library or genomic DNA from a blood sample of an AMD patient;

(2) screening the AMD DNA library or genomic DNA for sequences that hybridize under high stringency conditions to a wild type Ccl2 gene , Ccr2 gene; the ccl2 promoter, the Ccr2 promoter, or the Ccl2 enhancer and

(3) sequencing the sequences that hybridize to determine the identity of any mutations contained therein.

Claim 35. An expression vector comprising SEQ ID NO. 9.

Claim 36. An expression vector comprising SEQ ID NO. 10.

Claim 37. A method for screening for mutations that potentially cause or affect the development of AMD in a human comprising

(1) obtaining an AMD DNA library or genomic DNA from a blood sample of an AMD patient;

(2) screening the AMD DNA library or genomic DNA for sequences that hybridize under high stringency conditions to a wild type C5 receptor gene or C5a receptor gene;

(3) sequencing the sequences that hybridize to determine the identity of any mutations contained therein.

Claim 38. A method of treating or preventing AMD in a human comprising administering a nucleic acid encoding a wild type human Ccr2 gene, wild type Ccl2 gene or both to at least one eye of the human, wherein the nucleic acid is operably linked to a promoter, or a small molecule that promotes the function of human Ccr2 or Ccl2, and wherein said nucleic acid is expressed in at least one eye.